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Editorial

Redox regulation of inflammatory processes

Oxidative stress mediators such as Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) can be considered double edge molecules, whenever their role in inflammatory pathways is considered. Indeed, if on one hand ROS/RNS are important inflammatory effectors involved in the invading pathogens clearance and resolving the damage and inducing tissue repair; on the other hand, ROS/RNS can also be inflammatory initiators as a consequence of oxidizing biomolecules such as lipids, proteins and DNA, leading to tissue damage and switching acute to chronic inflammation.

The present special issue is aimed to bring new insights and discuss the current knowledge on the association between inflammation and oxidative stress and the redox signaling involved in inflammatory-associated diseases that can be identified by the word "*OxInflammation*".

OxInflammation represents the outcome of the cross-talk between oxidative stress (understood as increased ROS and RNS steady state concentrations) and inflammation. ROS/RNS and their related chemical species are efficient secondary messengers able to promote inflammatory responses by activating several transcription factors, (i.e. NF-κB, AP-1, c-Jun) and intracellular protein kinases (i.e. ERK, MAPK, INK and p38). The activation of inflammatory signaling pathways results in the release of cytokines, chemokines and other mediators that in turn, can further induce the production ROS and RNS by endogenous enzymes activation leading to a positive feedback loop, that can aggravate or even develop several diseases. This concept has been well described in the work by Percorelli et al. taking as example Rett syndrome, suggesting how redox posttranslational protein modifications can also be the mediators of pro-inflammatory processes. Similar pathways suggested for Rett syndrome have also been proposed for other two rare diseases, cerebral cavernous malformation (CCM) disease by Retta et al. and alkaptonuria by Braconi et al. The work by Retta et al. overviews the potential contribution of altered cell responses to oxidative stress and inflammatory events occurring locally in the microvascular environment, and consequent implications for the development of novel, safe, and effective preventive and therapeutic strategies, while the review by Braconi et al. highlights how proteome studies on inflammation and oxidative stress reveal the possibility to use inflammatory markers and oxidized proteins as biomarkers for alkaptnouria progression and possible treatments. Being mitochondria the main endogenous source of ROS, several articles have been focused on its role in the "oxInflammation" processes. The work by Rimessi at al. offers an overview on how inflammasomes are activated by mitochondrial mechanisms and the known role of mitochondrial ROS production in specific pathologies such as Alzheimer and Parkinson disease, and atherosclerosis. Lerner et al. focused on the role of mitochondria in exacerbating the inflammatory responses in chronic obstructive pulmonary disease (COPD) patients. The Special Issue is also characterized by a series of papers focused on the role of mitochondria in cardiac dysfunctions; such as the work by Alvarez et al., in which are summarized the current hypothesis of cardiac dysfunction related to energy metabolism and mitochondrial function in sepsis and endotoxemia models, introducing the important role of the lipid cardiolipin in cardiac energy management. Still on the cardiac functions modulated by inflammatory processes are the work by Perez et al. and Villamil et al.; in these papers, the possible protective role of thioredoxin-1 in myocardial dysfunctions is discussed. On the wave of the cardiac-dysfunction series there is also the work by Bombicino et al., where the association between mitochondrial dysfunction and cardiac function in diabetic animal model is described.

The neurological aspects of the cross-talk between ROS and inflammation are described by Neniskyte et al. showing data indicating that amyloid beta formation induces microglia phagocytosis of neurons via Protein Kinase C and NADPH oxidase activation. Of interest, the work by Castelazzi et al. in which is suggested that the decreased arylesterase activity of Paraoxonase-1 (PON-1) could be a common denominator of several neurological diseases. Voltan et al. clearly discusses the state of the art of the TNF-related apoptosis inducing ligand (TRAIL) pathway and redox signaling with special focus on vascular pathophysiology. Then there is the work by Ni et al., in which is described the role of oxidized lipids in melanocytes senescence.

The preventive role of polyphenols as anti-inflammatory molecules in obese animal model has been discussed by Bettaieb et al. The paper by Rocha et al. focuses on the different pathways involved in the interaction of dietary nitrate and polyphenols with gut microbiota and links this to inflammatory events, suggesting that the interactive cycles (nitrate-polyphenols-microbiome) could be a novel strategy for inflammatory diseases.

Finally, the work by Reynaert et al. provides a comprehensive overview on the role of RAGE in inflammatory signals and Magnani et al. focused its article on how exogenous sources of oxidative stress, such as fine particulates, thanks to the presence of transition metals are able to contribute to lung oxInflammation.

Overall, the papers presented in this Special Issue are able to confirm the role that oxidative stress and inflammation play in sev-

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eral pathologies, suggesting a possible common physiopathological mechanism.

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